SER. II, VOL. 11 May, 1949 No. 7

Q
11
.N529

TRANSACTIONS
OF
THE NEW YORK ACADEMY OF SCIENCES

Editor
ROY WALDO MINER

Associate Editor
PAUL LENIHAN

Contents

An Approximate Solution of Certain Diffusion Problems. *By* HOWARD REISS 236

The Response of the Mouse Adrenal Gland to Steroid Hormones: Report on Recent Investigation. *By* JAMES H. LEATHEM 239

Psychomotor Function as Correlated with Body Mechanics and Posture. *By* LULU E. SWEIGARD 243

Histoplasmosis in Animals. *By* C. W. EMMONS 248

Evidence for Pleistocene Man at La Jolla, California. *By* GEORGE F. CARTER 254

Conference Held: "International Conference on Vitamin E" 257

New Members 261

Copyright, 1949, by The New York Academy of Sciences

TRANSACTIONS of The New York Academy of Sciences, Series II, Volume 11, No. 7, May, 1949.
Entered as second-class matter December 12, 1947, at the post office at Baltimore, Maryland, under the act of August 24, 1912. This publication is distributed to members and is published monthly from November to June, inclusive, by The New York Academy of Sciences, Mt. Royal and Guilford Aves., Baltimore 2, Md. Editorial office: Seventy-Ninth Street and Central Park West, New York 24, N. Y.

Executive Director: EUNICE THOMAS MINER

*SECTION OF PHYSICS AND CHEMISTRY**

AN APPROXIMATE SOLUTION OF CERTAIN DIFFUSION PROBLEMS

By HOWARD REISS†

Introduction. This paper contains a description of two problems representative of those connected with the quantitative investigation of the procedures used to prepare monodispersed aerosols and hydrosols. The manner of solution of these problems will be indicated and the results will be discussed. The author hopes to create some interest in a field which just recently became quantitative.

A longer, more mathematical paper is planned for publication in the near future.

Aerosols and Spontaneous Nucleation. Monodispersed aerosols can be produced by the method of La Mer and Sinclair.[1] A uniform mixture of air, vapor, and condensation nuclei (small grains of foreign material) is cooled by passage through a column along which a temperature gradient is maintained. Upon the nuclear surfaces, the mixture cannot become supersaturated with vapor, and condensation results. Vapor from the internuclear regions diffuses to the surfaces and each nucleus is surrounded by a growing liquid droplet. The product is a monodispersed aerosol.

In order to maintain a high degree of monodispersity it is desirable for all the droplets to begin to form simultaneously. The occurrence of *spontaneous* nucleation which, at a later time, supplies nuclei additional to the foreign nuclei originally present is, therefore, to be avoided.

These nuclei, formed spontaneously, are merely fragments of the condensed phase which appear in large numbers when the degree of supersaturation exceeds a certain critical limit. Becker and Boring[2] have discussed the phenomenon quantitatively in terms of a *supersaturation* ratio: the ratio, at a given temperature, of the vapor pressure in the supersaturated state to that in the saturated state. The critical ratios, signalizing the onset of spontaneous nucleation, calculated by these authors, are in good agreement with experiment.

If the air-vapor-nuclei mixture, previously mentioned, is cooled rapidly, it is conceivable that the diffusion process will not be able to drain vapor from the internuclear regions fast enough to prevent the supersaturation ratios for these regions from exceeding the critical limit. Whether or not this will happen can only be decided when the diffusional boundary value problem has been solved so that the concentration of vapor in these regions can be computed at every instant.

The Growth Rate of Sulfur Hydrosols. Monodispersed sulfur hydrosols can be produced by the method of La Mer and Barnes.[3] Dilute solutions of acid and thiosulfate are mixed, whereupon the following reaction occurs

$$2HS_2O_3^- = 2S + 2HSO_3^-.$$

At first, the sulfur is released in solution. As the reaction proceeds, the solution becomes supersaturated with respect to sulfur and a critical limit is reached. At this point, spontaneous nucleation occurs, and the sulfur in solution diffuses towards the newly formed sinks. If the diffusion process is sufficiently rapid, it will remove dissolved sulfur more rapidly than the chemical reaction can produce it, and the concentration will be lowered below the critical limit, bringing an end to the nucleation process. During the final stage, the sulfur droplets

* The Section of Geology and Mineralogy held its meeting on April 4, 1949, at which Ellis A. Johnson presented a paper, illustrated with lantern slides, entitled *The Magnetization of the Earth's Crust*. This will be published in a later issue of TRANSACTIONS.

† Department of Chemistry, Columbia University, New York, N. Y. This paper was presented at the meeting of the Section on April 5, 1949.

grow by means of diffusion in the field of a uniformly distributed source, *i.e.* the chemical reaction.

It is of interest to know whether or not the reaction is catalyzed at the droplet surfaces, *i.e.* can the growth of the droplets be ascribed partly to deposition from an accelerated reaction at their surfaces, or is it simply describable in terms of the diffusion of molecularly dispersed sulfur?

Here, again, a decision can only be arrived at by the computation of the droplet growth rates upon the basis of a diffusion mechanism and by comparing the result with experimental data.

Methods of Solution. The aerosol and hydrosol problems, described previously, contain basic elements of similarity. Both involve diffusion towards many randomly located sinks (nuclei), and both are peculiar in that a boundary, namely the droplet surface, is not geometrically fixed but moves as the droplet grows. Because of these facts, the problems cast into their natural forms are too difficult to solve. It is, therefore, expedient to replace them by artificial problems which retain the important features of the natural ones, but which are amenable to solution.

Instead of considering a large number of nuclei, it is practicable to confine attention, in each case, to the neighborhood of a single nucleus, and regard this neighborhood to be spherically symmetric. The presence of the other nuclei, which compete for the diffusing material, can be accounted for by enclosing the neighborhood in an impermeable spherical shell of radius h, centered at the nucleus whose radius is x. The impermeability of the shell prevents the material beyond its confines from depositing on the central nucleus, so that, in effect, this material may be considered to condense on the competing nuclei. The magnitude of the radius h is chosen so that the volume contained in the shell equals the reciprocal of the number density of the nuclei, *i.e.* the effective volume of the diffusion field belonging to each nucleus.

The spherical symmetry renders it sufficient to determine position by the magnitude, r, of the radius vector originating at the center of the nucleus of radius, x.

In the aerosol case, the initial concentration of vapor c_o, corresponding to the temperature, T_o, is everywhere uniform. The boundary condition at x corresponds to the maintenance, there, of the saturation pressure.

In the hydrosol case, c_o represents the concentration of sulfur molecules when the critical limit of supersaturation is reached. The boundary condition at x corresponds to the maintenance of saturation, *i.e.* the concentration at x is always equal to the solubility of sulfur. In the hydrosol case, the diffusion equation includes a source term, corresponding to the ever-present chemical reaction.

In both cases the movement of the boundary at x makes it impossible to use standard methods to solve the diffusional boundary value problems. Ordinarily, the equation governing the diffusion phenomena is

$$\frac{\partial^2 c}{\partial r^2} + \frac{2}{r}\frac{\partial c}{\partial r} = \frac{1}{D}\frac{\partial c}{\partial t} - \frac{k}{D}, \qquad (1)$$

where c is the concentration of the diffusing species. This equation is valid, provided that Fick's law[4] holds. D is the diffusion coefficient, t, the time, and k is the source function equal to zero in the aerosol case. In certain physical situations, the flux of material through any surface in the diffusion field is much greater than the rate of change of concentration on that surface. In such cases, (1) can be replaced by the approximation

$$\frac{\partial^2 c}{\partial r^2} + \frac{2}{r}\frac{\partial c}{\partial r} = -\frac{k}{D}. \qquad (2)$$

EQUATION (2) is equally simple to solve, independently of whether the boundary does or does not move. It happens that (2) is admissible whenever x/h is small. The simplifying feature in the problem involving (2) is the fact that the time dependence of c is not determined by (2), but, rather, by the boundary conditions.

The condition for an impermeable shell at h is ordinarily

$$\left(\frac{\partial c}{\partial r}\right)_{r=h} = 0 \qquad (3)$$

The only solution of (2), compatible with (3) is the trivial solution

$$c = c(t) \qquad (4)$$

which implies that c does not depend upon r but only upon t. It is, therefore, necessary to replace (3) by a more reasonable condition.

This can be done by realizing that the impermeable shell limits the amount of material available for diffusion. Consequently, the amount of material diffusing into the surface at x represents, exactly, the amount of material lost from the region between the boundaries at x and h. The maintenance of this balance can be worked into a condition suitable for the replacement of (3).

The rate at which x changes is, of course, determined by the process of diffusion. It is given by

$$\frac{dx}{dt} = \frac{WD}{A\rho}\left(\frac{\partial c}{\partial r}\right)_{r=x} \qquad (5)$$

where W is the molecular weight of the diffusing species, A is Avogadro's number, and ρ is the density of the droplet.

Results. The solution of the aerosol problem yields the value of $c\ (r, t)$. This value will always be highest at $r = h$, since diffusion occurs towards the sink at x. Consequently, if spontaneous nucleation is to take place, it will take place at h, first. It was found convenient to define a *cooling* ratio

$$c(h, t)/c(x, t). \qquad (6)$$

Since $c\ (x, t)$ is the saturation concentration, spontaneous nucleation would be predicted whenever (6) exceeded the critical ratio computed by the method of Becker and Doring. It was useful to plot both the critical ratio and cooling ratio against time (temperature) on the same graph. The intersection of the two curves predicts spontaneous nucleation.

Calculations and plots of these types were made for naphthalene aerosols which happen to be similar to a number of sols prepared in this laboratory. The calculations were carried out for different cooling rates, different concentrations of foreign nuclei, and different values of T_o. The results indicate that so long as there are more than 100 nuclei per cc. whose surfaces cannot support supersaturation, spontaneous nucleation cannot occur during the preparation of a naphthalene-like aerosol, even when the cooling rate is ten times normal, *i.e.* 530° K./sec.

The aerosols produced in this laboratory contain about 10^6 nuclei per cc. The comparative ease with which a monodispersed aerosol can be produced supports the above conclusion.

The solution of the hydrosol problem yields the dependence of x upon time. Zaiser and La Mer,[5] using light scattering methods, have measured this dependence. They plotted four curves (corresponding to different conditions) of x^2 against time.

The theoretical functional relationship is

$$x^2 = f(t, W, \rho, k, h, D, c_0). \qquad (7)$$

Both D and c_o were not accurately available. However, c_o has been estimated by spectrophotometric means[6] to lie in the range

$$3\text{–}4 \times 10^{-6} \text{ g. atom sulfur/liter.} \qquad (8)$$

Furthermore, if it is assumed that the diffusing unit is S_8, D should lie in the range

$$4\text{–}7 \times 10^{-6} \text{ cm}^2/\text{sec}, \qquad (9)$$

since all molecules which compare in size with S_8 have diffusion coefficients lying in this range, in aqueous solution at room temperature.[7] Equation (7) could be nicely fitted to the experimental curves if c_o and D were chosen, respectively, to be

$$c_0 = 4.7 \times 10^{-6} \text{ g.atom sulfur/liter} \qquad (10)$$

$$D = 2.0 \times 10^{-6} \text{ cm}^2/\text{sec}. \qquad (11)$$

These values compare well with (8) and (9) estimated by independent means.

It is to be concluded that the suggested diffusion mechanism for the growth of sulfur droplets upon which equation (7) is based provides an adequate quantitative description of the observed phenomena.

Summary. An approximate method is described for treating problems of diffusion, complicated by moving boundaries and the presence of many competing sinks.

This method is used to investigate problems of the growth of monodispersed sols.

One conclusion is that in a naphthalene-like aerosol system, growing by the diffusion of vapor towards sinks (nuclei) which cannot support supersaturation, spontaneous nucleation will not occur if the number density of nuclei exceeds 100 per cc.

Another conclusion is that the growth of sulfur hydrosols prepared by the method of La Mer and Barnes is not complicated by a surface catalyzed reaction, but may be explained on the basis of a simple diffusion mechanism.

BIBLIOGRAPHY

1. La Mer & Sinclair. OSRD No. 1857, Dept. of Commerce, Office of Publication Board, Report No. 944. (Also to appear in Chem. Rev. 1949.)
2. Becker & Doring. 1935. Ann. Physik **24:** 719.
3. La Mer & Barnes. 1946. J. Coll. Sci. **1:** 71, 79.
4. Longsworth. 1945. Ann. N. Y. Acad Sci. **46:** 212.
5. Zaiser & La Mer. 1948. (December) J. Coll. Sci. Figure 3.
6. Zaiser & La Mer. 1948. (December) J. Coll. Sci.
7. I. C. T. **5:** 63–76.

SECTION OF BIOLOGY

THE RESPONSE OF THE MOUSE ADRENAL GLAND TO STEROID HORMONES: *Report on Recent Investigation*

By JAMES H. LEATHEM*

An adrenal x-zone is present in most strains of mice and lies between the permanent cortex and the medulla of the adolescent male and female, and the nulliparous female.[1,2] Normally, the x-zone of the male disappears about the fifth week, whereas, in the virgin female, it persists for an estimated 80 to 200 days. Pregnancy, however, incites a rapid disappearance of the adrenal x-zone. The tissue of this zone can be identified easily, as the cells are eosinophilic and in cords which interdigitate with the medulla, but, as and when the zone disappears, the interdigitation is lost and a connective tissue capsule forms between the cortex and medulla.

The physiological mechanisms of pregnancy include a marked increase in secretion of estrogen and progesterone. Only during pregnancy is the mouse subjected to a continuous secretion of progesterone and, therefore, this steroid may be responsible for the adrenal x-zone disappearance. In fact, Selye[3] reported x-zone disappearance when 3 mg. of progesterone were injected daily for 20 days, but other investigators, using smaller dosages, but adequate to support pregnancy in spayed mice, failed to support these results. Since large doses of progesterone may have an androgenic action, this may explain Selye's results, and, in our studies, 0.25 mg. of progesterone on alternate days for a month had no effect on the x-zone.

Adrenal excitation may occur during pregnancy and, consequently, secretions from this gland may be altered in such a way as to influence its histology. Indeed, Selye[3] reported x-zone disappearance with desoxycorticosterone acetate (DCA). In our experience, the adrenals of immature female mice are not altered by 0.25 mg. of DCA daily for 30 days or by 1.0 mg. of the steroid daily for 20 days.

* Department of Zoology and Bureau of Biological Research, Rutgers University, New Brunswick, New Jersey. This paper, illustrated with lantern slides, was presented at the meeting of the Section on April 11, 1949.

Although estrogen is secreted during the estrus cycle, the level of secretion during pregnancy may be altered sufficiently to elicit x-zone disappearance. A review of the action of administered estrogen[4] favors the view that steroids with estrogenic activity do not influence the x-zone. In our laboratory, stilbestrol, estradiol, estradiol dipropionate, and estrone at different dosage levels failed to alter the x-zone of the immature female, but did induce a ring of cytolysed cells to appear in the neighboring fasciculata. Administration of 2 μg. of stilbestrol daily for 16 days to 24 day old male mice at the start prevented the normal disappearance of the x-zone, presumably, by blocking maturation of the reproductive system.

Frequently, a combination of hormones is more beneficial than one alone and so the combination of estrogen and progesterone was injected into female mice, but adrenal x-zone histology was not influenced.[5]

Since the x-zone tissue disappears in the male mouse concomitant with sexual maturity, it was soon learned that castration prevented this adrenal change. What's more, testosterone propionate as replacement therapy or when administered to normal or spayed female mice quickly deletes the x-zone.[6] Recently, Jones[7] extended these results to include the hypophysectomized castrated mouse. From the accumulated data, the response of the x-zone tissue seems to be specific for androgens and, to date, we have eliminated this adrenal zone in immature female mice with testosterone propionate, methyl testosterone, vinyl testosterone, and androsterone.* These findings prompted us to study the x-zone response to testosterone propionate in assay fashion and compare its sensitivity with that of seminal vesicles and to evaluate the activity of other steroids on this end organ. Seminal vesicle weight response was determined in 22 day old male mice at 72 hours after a single injection of testosterone propionate. Fifty μg. of the steroid induced a 100 per cent increase in seminal vesicle weight and served as a good indication of sensitivity (TABLE 1). For the adrenal x-zone study, 30–32 day old female mice were used and the subcutaneous and intraperitoneal routes of administration investigated. A single injection of 0.5 mg. of testosterone propionate removed the x-zone by day 7 when injected subcutaneously, but no effect was observed after the intraperitoneal injection. Thereafter, testosterone propionate was administered as a single subcutaneous injection and mice killed 7 days later. The adrenals were fixed in Bouin and stained with hematoxylin and eosin. Dosages of 0.05, 0.1, 0.2, 0.3, 0.4, 0.5, 1.0, and 2.5 mg. were studied with the 0.1 mg. dosage proving to be the critical level under our experimental conditions. At the 0.1 mg. level, the x-zone had disappeared or was clearly influenced in all cases, whereas the 0.05 mg. dose did not cause x-zone disappearance. All of the higher dosages abolished the x-zone tissue (TABLE 2).

TABLE 1

Response of immature mice to a single subcutaneous injection of testosterone propionate acting for 72 hours

Treatment	*No. of mice*	*Body wt. g.*	*Testis mg.*	*Seminal vesicle mg.*
Control	30	11.3	76	6.2
0.025 mg.	15	10.3	61	9.6
0.050 mg.	12	12.5	74	13.9

TABLE 2

Effect of a single subcutaneous injection of testosterone propionate on the female mouse x-zone after 7 days

Dosage mg.	*Condition of x-zone*	*Alkaline phosphatase*
None	present	+
0.05	present	0–+
0.10	absent or small	0–+
0.20	absent	+
0.50	absent	++
1.00	absent	
2.50	absent	++++

Recently, Elftman,[8] using the histochemical technique for alkaline phosphatase, observed this enzyme in the cortex of the adult male, whereas the female gland or those

* The androgens were generously supplied by Ciba Pharmaceutical Products, Summit, New Jersey.

adrenals from immature mice were essentially devoid of the enzyme unless treated with testosterone propionate. This interesting finding suggested another mouse adrenal relationship to androgenicity, and a correlated study of x-zone disappearance and alkaline phosphatase appearance was made. Thirty-day-old female mice were given single subcutaneous injections of varied doses of testosterone propionate (Perandren, Ciba). One adrenal was fixed in Bouin and the other in cold 80 per cent alcohol for the enzyme. The x-zone disappearance correlated with the previous study and required 0.1 mg. of the steroid, whereas 0.5 mg. was needed for a definite enzyme increase. Elftman[8] reported an increase in alkaline phosphatase when the same steroid was injected on two successive days in 0.1 mg. daily amounts and the mice killed on day 3. A repeat of this experiment in female mice confirmed an enzyme increase, but x-zone disappearance was complete in only 3 of 7 mice. The problem of time and repeated dosages is under further study.

Korenchevsky and Hall[9] observed an androgen antagonism in hyperthyroidism and these data suggested that similar results may be obtained using the x-zone as the end point. Accordingly, hyperthyroidism was induced in 22-day-old mice by putting them on a diet containing 0.1 per cent thyroglobulin (Proloid) for 17 days. The testosterone was administered as a single injection when the mice were 32 days old. In the hyperthyroid state, a 0.2 mg. dose of androgen did not cause x-zone disappearance, but 0.5 mg. was effective showing that the effective dose level of androgen was clearly above the dosage needed in normal mice.

One of the perplexing aspects of the mouse adrenal problem is the disappearance of the x-zone in female mice following treatment with gonadotrophins.[7,10] In our experience with chorionic gonadotropin, pregnant mare serum and rat anterior pituitary injected for 20 days, we have seen x-zone disappearance in the female, but not as a consistent response, despite uniform treatment. Pfeiffer and Hooker[11] have studied the source of ovarian androgen in the mouse and obtained androgen release under pregnant mare serum stimulation. Thus, ovarian androgen might be responsible for x-zone disappearance under gonadotrophin stimulation. Argyris in our laboratory, in preliminary studies, has found an increase in adrenal alkaline phosphatase along with x-zone disappearance when chorionic gonadotrophin was injected for 20 days. The increase in enzyme was not seen if the x-zone remained, despite identical treatment.

Since the androgen-adrenal x-zone relationship is tenable, experiments involving the reproductive system have included a study of the adrenal as a measure of hormone secretion in addition to seminal vesicle weight. For example, injection of 2 μg. of stilbestrol daily for 16 days into 24-day-old male mice results in suppression of the reproductive system and retention of the x-zone. Similarly treated mice were then studied at 10, 20, and 30 days after the last injection to observe the rate of recovery. The testes contained spermatozoa 20 days after the last stilbestrol injection, and seminal vesicle weight was normal, but the x-zone was still present. Ten days later, the x-zone had also disappeared (TABLE 3). Normally, spermatozoan appearance occurs at the same time as x-zone disappearance.

TABLE 3

Recovery of 24 day old male mice following 0.002 mg. stilbestrol × 16

Treatment	*No.*	*Testis*	*Seminal vesicles*	*Adrenal*	*x-zone*
S + 10 days	7	83	18	7.9	+
Control	6	178	116	5.7	—
S + 20 days*	7	144	114	5.0	+, —
Control	7	183	117	4.2	—
S + 30 days	7	180	220	4.5	—
Control	6	200	176	4.0	—

* 5 I.U. PMS daily in recovery period hastens x-zone disappearance.

The relationship of age and hormone responsiveness has been of interest to us for some time and, again, the adrenal x-zone has served as an adjunct to studies on the reproductive system. The responsiveness

of mice 5, 10, 15, and 20 days old to a single subcutaneous injection of 50 I.U. of pregnant mare serum (PMS) was determined five days later. In the male, seminal vesicle weight responded maximally in mice 10 days old at injection. At this age and older, the adrenal x-zone disappeared as the testis steroid secretion was prematurely augmented.[12] The five day old male, too, responded to PMS as indicated by seminal vesicle weight increase, but the adrenal x-zone was not influenced at 5 to 15 days after one injection, suggesting that the tissue cannot respond at this age or that insufficient androgen was released. In the female, the reproductive system responded at all above mentioned ages, but the x-zone remained intact.

A critical age in rat development seems to exist approximately 10 days postnatally. Merklin[13] has found that as little as 1 μg. of estradiol dipropionate injected into 10 day old mice as a single injection will clearly delay the appearance of spermatozoa in the testis and that adrenal x-zone disappearance is related to recovery of testis function.

Ten day old mice of both sexes have also been studied after the single subcutaneous injection of 1 mg. of testosterone propionate. A study of the reproductive system and adrenal was made at 10, 20, and 30 days after the injection. In male mice, the androgen delayed the onset of spermatogenesis slightly, but full recovery was observed in 30 days. The x-zone was removed by the testosterone injection and the subsequent recovery of the reproductive system kept a continuous androgen outpouring so that it did not return. In the female, the reproductive system was retarded and the x-zone was lacking for 20 days after injection, but, 30 days after hormone treatment, the x-zone had reappeared (TABLE 4).

Other studies have related brown degeneration in the mouse adrenal to tumors.[7,14] It was deemed of interest to study the female mouse adrenal in precancerous periods for possible adrenal changes. The experiment involved feeding 0.1 per cent 2-acetylaminofluorene in a semisynthetic diet containing 24 per cent casein for periods of 45 and 100 days, during which time hepatic tumors did not form. Adrenal histology was normal in all mice examined at the 45 day period, but, at 106 days, the adrenal x-zone in most of the control mice had small areas of vacuolization suggestive of beginning degeneration. No x-zone degeneration was observed in the adrenals from mice fed the carcinogen. The adrenals were also studied for alkaline phosphatase, which, in the normal, is distributed in the capsule, zona glomerulosa and in capillaries, and the drug fed animals exhibited this normal pattern. The control adrenal glands exhibited a definite alkaline phosphatase concentration in the x-zone, specifically, in the nuclei of degenerating cells. Since the factors inciting degeneration of the x-zone in female mice of 80 to 200 days are not known, the effect of carcinogen feeding is not explainable at this time.

TABLE 4

Testosterone propionate (1 mg.) given in one injection to 10 day old mice

Treatment	*Testis*	*Seminal vesicles*	*Ovary*	*Uterus*	*Adrenal x-zone* ♂	♀
T.P. + 10 days	42	21	3.0	14	−	−
Control	65	6	3.4	13	+	+
T.P. + 20 days	99	37	3.6	43	−	−
Control	134	27	4.2	35	−	+
T.P. + 30 days	147	92	6.0	66	−	+
Control	159	100	6.0	74	−	+

+ = present
− = absent

BIBLIOGRAPHY

1. MASUI, K., & Y. TAMURA. 1926. The effect of gonadectomy on the structure of the suprarenal glands of mice, with special reference to the functional relationship between this gland and the sex gland of the female. J. Coll. Agric. Imp. Union. Tokyo. **7:** 353.
2. HOWARD-MILLER, E. 1927. A transitory zone in the adrenal cortex which shows age and sex relationships. Am. J. Anat. **40:** 251.
3. SELYE, H. 1940. Compensatory atrophy of adrenals. J.A.M.A. **115:** 2246.
4. LEATHEM, J. H., & S. SILVERMAN. 1945. Action of stilbestrol on the female mouse adrenal gland. Proc. Penna. Acad. Sc. **19:** 60.
5. WARING, H. 1942. Effect of hormones on

degeneration of the x-zone in the mouse adrenal. J. Endocrinol. **3:** 123.
6. STARKEY, W. F. & E. C. H. SCHMIDT, JR. 1938. The effect of testosterone propionate on the x-zone of the mouse adrenal. Endocrinol. **23:** 339.
7. JONES, I. C. 1948. Relationship of the pituitary to the x-zone of the mouse adrenal. Proc. Soc. Exper. Biol. & Med. **69:** 120.
8. ELFTMAN, H. 1947. Response of the alkaline phosphatase of the adrenal cortex of the mouse to androgen. Endocrinol. **41:** 85.
9. KORENCHEVSKY, V. & K. Hall. 1941. Correlation between sex hormones, thyroid hormone and desoxycorticosterone as judged by their effects on the weights of organs of gonadectomized rats. Biochem. J. **35:** 726.
10. MCPHAIL, M. K. & H. C. READ. 1942. The mouse adrenal. Anat. Rec. **84:** 51.
11. PFEIFFER, C. A. & C. W. HOOKER. 1942. Experiments on the source of ovarian androgen in the mouse. Anat. Rec. **83:** 543.
12. BISHOP, D. H. & J. H. LEATHEM. 1946. Response of prepuberal male mice to equine gonadotropin. Anat. Rec. **95:** 313.
13. MERKLIN, R. J. (Unpublished)
14. TOBIN, C. E. & J. P. BIRNBAUM. 1947. Some factors influencing brown degeneration of the adrenal gland in the swiss albino mouse. Arch Path. **44:** 269.

SECTION OF PSYCHOLOGY

PSYCHOMOTOR FUNCTION AS CORRELATED WITH BODY MECHANICS AND POSTURE

By LULU E. SWEIGARD*

The term psychomotor, separated into its parts, means mind and movement. It suggests that activity of the mind is accompanied by movement in and of the human body. Movement, initiated in the body itself, is produced by the action of muscles, mainly on bones. Hence, we may say that activity of the mind is accompanied by muscle action, or that thinking has its motor components. Experimental work substantiating such statements has been done mainly in the field of Psychology, since the time of William James, who first placed emphasis on the theory.

Because of preconceived and frequently inaccurate ideas regarding body mechanics and posture, especially posture, the meaning of each, as used here, must be defined. Consider body mechanics first. Physics defines mechanics as that branch which treats of the motions of bodies and the causes of changes in these motions. Body mechanics, then, must be defined, similarly, as treating of the movement of the human body and the causes of changes in its movement. Causes of changes in motion of the human body are no different from the causes of changes in motion of inanimate bodies. Hence, we cannot logically escape the fact that physical laws apply to the balance and movement of the human body, just as to any other structure. Failure to recognize this fact has led to illogical and unscientific reasoning regarding body alignment and movement. Further, it has decreased the value of a number of experimental studies by its interference with a scientific approach to problems.

Consider next the term, posture. Some authors speak of "postures," thus implying that each individual can have more than one posture. In view of the results of experimental work, this is not true. Perhaps the erroneous idea that a person can have "postures" is due to failure to distinguish between the two terms, posture and position. Position means the manner of being placed or arranged, as in standing, sitting, or stooping. Posture, in contrast, means the *relative* position of the various parts of anything. In the human body it is the relative position, or pattern of relationship, of the parts of the body. A pattern of relationship of body parts, or posture, tends to exist in each and every position the body may assume.

The meaning of posture may be clarified further by turning to the results of research on body alignment. One study was made by means of radiographs of the skeletal framework in the standing position; an-

* School of Education, New York University, N. Y. This paper was presented at the meeting of the Section on April 18, 1949.

other study was made by means of photographs of the nude body, likewise in the standing position. Each of these studies indicates: (1) that in every individual there is a consistent and persistent pattern of relationship in the alignment of the various parts of the body as its equilibrium is maintained in the upright position; (2) that there is similarity in the pattern of alignment of the majority of individuals; and (3) that the nearer the parts of the body are to its center of gravity, the less variable the pattern of relationship of parts tends to be. The latter is logical from the standpoint of physical laws.

The posture pattern also influences the manner in which all movement activities are performed. To some extent this can be verified by observation. Characteristic postural idiosyncracies are noticeable in friends whether they are standing, sitting, or moving. The degree of influence, however, of the postural pattern on movement must be determined through experimental study. One such study of the influence of the posture pattern on the movement of walking will be completed in the near future.

Having defined body mechanics and posture, there is need for consideration of *how* ability to maintain equilibrium and to move is established and *why* patterns of postural alignment and movement tend to be consistent and persistent, even to grow worse. The human being is a self-directing mechanism through the action of his muscular system on his skeletal framework. Muscles, however, do not contract to produce movement unless and until they receive motor messages from the central nervous system. How are these messages instigated? What determines their muscle destiny? Is the motivating force a thought or idea? If so, is the thought concerned with muscle action; or is it concerned with movement only?

To answer such questions briefly, the development of ability to move must be considered. The body is the only means of expressing one's self. For expression there must be movement. Expression is sought from birth, and movement, to accomplish this, must be learned slowly by trial and error methods until adequate muscle coordination is established.

The individual is born with certain primary reflexes which he is unable to control, or change but briefly, if at all. Beyond primary reflexes, other reflexes make it possible at birth to move arms and legs and to squirm the trunk, but without specific direction. Neuro-muscular coordination toward directed and effective movement has not yet been established. But it can be and is, in the early years of life. The learning process takes place through building S-R bonds—muscle responses to specific stimuli. These are slowly established and, more frequently than not, the initial motivating force which activates many and complicated S-R bonds is the idea or thought of movement. Thus neuro-muscular action patterns are conditioned so that movement can be directed effectively toward the accomplishment of purpose.

Many influences play on this conditioning process: inherited structure, especially skeletal, muscular, neurological; mechanical, such as toys, furniture, clothing; pathological, as disease or sickness of any kind; injury, even though it thwarts only temporarily the ability to move; nutrition, with its general effects on growth and health; sociological, in its adjustment to others of society; and psychological, which are of great variety. Within the realm of psychological influences occur some of the most detrimental. The child imitates the posture and movement of those he admires and loves. He unconsciously absorbs and tends to live imposed ideas about posture and movement, whether they are good or not. He is influenced by the emotional stability of his parents, teachers and others about him. Discipline, or its lack, also has an important influence on the degree of muscular strain created in his body.

Thus, through trial and error methods, influenced by innumerable factors, neuro-muscular reflexes concerned with balance and movement of the body are conditioned;

they become a matter of habit. Habits exist in the neural pathways traveled by motor messages through and from the central nervous system to the muscles. Continued use over the years sets more firmly the habit patterns of neuro-muscular action. Beyond their use required by the usual activities of life, few people are fortunate enough to escape the habit-setting effects of time-honored postural admonitions and of exercises for different parts of the body. As they are practiced, conditioned reflexes are used, habits are intensified, and muscular strain is added.

It may be concluded then that conditioned neuro-muscular action accounts for and is the *main cause* of the consistence and persistence of a postural pattern and manner of movement; also, that it limits both the ability to assume a good posture at will and the ability that may be attained in the performance of a skill, whether this skill be one used in work, play, or recreation.

With an understanding of the reason for postural pattern and manner of movement, attention can be turned to the possibility of their change, whether change is worth while, and how it can be brought about.

No one can deny the fact that that which has been conditioned in the human being can be reconditioned. Just as surely as Pavlov conditioned dogs so that their mouths watered at the sound of a bell, so can the human being be reconditioned in his muscular responses to stimuli. Toward what end, is the important consideration.

To determine logical and worth-while goals in the reconditioning process, reasoning should proceed along mechanical lines. The established goal which has led to the construction of increasingly better inanimate mechanisms is a greater degree of efficiency—a better ratio of useful work to energy used. Granting that this is a worthy goal in engineering, economically and otherwise, the important factors influencing efficiency in inanimate structures should be noted. They are, primarily, a well-balanced supporting framework, ease of movement of parts of the structure in the performance of their work, and movement of all parts in accord with physical laws and principles of mechanics.

The fundamental aim in education and training for better body mechanics and posture likewise should be a greater degree of efficiency. This aim cannot be attained in the function of the human body without consideration of the same primary factors which influence the degree of efficiency in other mechanisms. In terms of the human body they are: alignment of the supporting skeletal framework in accord with principles of balance; flexibility of all joints in accord with their architecture and with principles of muscle function; and good mechanical use of all parts of the body, especially of the upper and lower extremities.

Before any procedure can be employed effectively to increase efficiency in the operation of the human mechanism, one must have (1) a clear concept of *ideal* body alignment, and (2) knowledge of deviations from the ideal which are typical of the majority of individuals.

To consider the ideal first, alignment of the skeletal structure would conform in so far as its architecture would allow, with the simple principles of balance. These principles are concerned with the adequacy of the various bases for support of weight throughout the structure, and the distribution of the weight of structure so that it is near the central line and near the base. Conformity with these principles determines the degree of stability of equilibrium. Logically, the true base of the human structure is the pelvis, since the center of gravity is located in that area. Recognition of this is essential to scientific reasoning in regard to both body alignment and movement.

As the concept of good skeletal relationships and alignment is built, accompanying muscle action and body contour can be studied and interpreted in terms of the internal framework. The latter is simple as a statement; actually, it is difficult and complicated—never ending and never fully known. It must be taken into consideration, too, that structural builds are different;

hence, body contours are different, even when bones are well balanced. Esthetic preference of a type of human figure must not be allowed to confuse scientific thinking.

Study of the deviations from ideal alignment indicates that there is, in general, a typically poor posture pattern found in the majority of individuals. The familiar postural difficulties, as forward head, round back, prominent abdomen, etc., should be interpreted in terms of poor skeletal relationships. In the majority of individuals, to a greater or less degree, interpretation shows that the following faulty relationships tend to occur. The trunk is too short in back, too long in front, and asymmetrical laterally. The lower extremities assume a backward and laterally different slant toward the feet. There are few, if any, spinal columns without lateral deviations of more or less degree, and few whose natural forward-backward curves are well balanced for weight support. Both the ribs and the shoulders are invariably supported at a higher level from the pelvic base than their bony attachments indicate. Finally, but not to complete the picture, the head tends to be too far forward, as well as rotated and tipped laterally.

All these deviations from good alignment imply a pattern of overwork, or hypertonicity, in muscle action. It must be so, since individuals, by means of muscle action, maintain their equilibrium in a consistently poor pattern of alignment.

As a concept of typically poor skeletal alignment is built, relative muscle action and distortion of body contour occurring with it must be interpreted. There are a few experimental studies which aid in this. They do not give a solution, however, to the intricate problem of muscle action; they serve mainly to point the way to better reasoning on a problem which seems unending in its complication.

Posture teachers are indebted, primarily, to neurologists and psychologists for the scientific support of changes now occurring in their teaching. Such changes are supported by the principle: thinking influences muscle action. It can influence it detrimentally or beneficially. Its detrimental influence is within the experience of most people, notably that of continuing the work and battles of the day after going to bed at night, thus building greater strain in the body instead of releasing it. Its beneficial influence is demonstrated especially in the work of Mabel Elsworth Todd* and Sister Kenny, and to some degree in the work of many others in both Medicine and Education.

Mental activity, then, especially that in which the thought of movement is involved, is the most effective means employed thus far to recondition neuro-muscular action patterns in the body. Such mental activity can be encouraged in a variety of ways. The key to each, however, is mental imagery, that is, visualization of an imaginary situation. Teaching can and should proceed in a manner which will lead the student to think in terms of concrete images. As this is achieved, image-directed muscular responses tend to occur. When no voluntary aid is given by the subject, concentration on a mental image promotes a coordination of muscle action whose resultant is the line of action of forces at play in the imagined situation.

In order to obtain the best results, especially when working with a group, the teacher should first help students gain the ability to change muscle action through concentration on mental imagery, without giving voluntary aid, and to recognize and interpret the kinesthetic experience accompanying such change. As this ability is attained, voluntary movement may be added to promote greater power in the newer muscle patterns and to speed up change. Since time does not allow detailed consideration of movement, suffice it to say that it must be carefully chosen, and that its effectiveness is controlled to a large extent by simultaneous use of appropriate mental imagery.

Of great importance to effective results in teaching are the answers to the following

* Todd. M. E. *The Thinking Body.* Paul B. Hoeber, New York.

questions. What mental images shall be used, where in the body, and what shall be the direction of visualized movement? The answers are determined from the typical deviations of skeletal structure from ideal alignment, and from the principles governing muscle action.

Through comparison of typically-poor with ideal skeletal relationships the writer determined eight "lines-of-action" needed to produce better body alignment and greater freedom of movement. Each line-of-action extends from one part of the skeleton to another. Along each line, distance between bones either increases or decreases. Such change is promoted by visualized action, without voluntary movement. The mental images on which the individual concentrates to promote the line-of-action are so designed that the resultant of the action in the image will be identical with the desired line-of-action. It may require recoordination of as many as fifty or one hundred muscles to produce this resultant.

Briefly, the lines-of-action, stated according to their purpose and direction of movement, are as follows: (1) to lengthen the back downward; (2) to widen across the back of the pelvis between the great trochanters of the thighs; (3) to narrow across the front of the pelvis; (4) to produce alignment of the knee with the thigh joint; (5) to shorten the inside of the foot toward the heel; (6) to narrow the rib-case; (7) to shorten from the midfront of the pelvis to the top of the forward lumbar curve; and (8) to shorten (or lengthen) from the top of the breast bone to the top of the forward cervical curve.

Thus, the lines-of-action determine the location and direction of movement in the body, which is only to be visualized. Images that may be used to promote lines-of-action are many and variable. However, they should seldom, if ever, be created in terms of muscle action which is so highly complicated. Furthermore, the student does not need actually to know a thing about muscles to change his posture and movement.

Presenting movement to be visualized is not the only way to promote thinking in terms of mental images. Another way lies in the educational procedure itself—the presentation of pertinent facts in an interesting and challenging way, leading the student to think in terms of concrete imagery in his own body. It should be remembered, also, that people tend to be visual, kinesthetic, or auditory minded. This requires some modification in procedure to make it equally effective for all.

To summarize the primary qualifications of one who might be termed an engineer of the human body, they include the following: a clear mental picture of ideal skeletal alignment, supported by knowledge of physical laws and principles of mechanics; ability to visualize within body contour the existing faulty alignment of bones and to interpret the interrelationship between this and existing muscle action; knowledge of change needed in bony relationships to promote greater structural stability and freedom of movement; and finally, logical aims by which proposed change in sleletal alignment may be evaluated.

To avoid possible misinterpretation of some of the statements that have been made, it seems advisable, before completing this discussion, to clarify the difference between two terms, efficiency and relaxation, which too often are used interchangeably.

Dr. Edmund Jacobson, in his report*, states "muscular relaxation (conceived as a limit) can be regarded as freedom from effort, physical and mental." If this definition is accepted, there is only one way to attain relaxation to the greatest degree, and that is during rest of the body in a specified lying position and with minimum mental activity. Increased efficiency, on the other hand, means the accomplishment of purpose in any activity and in any position with the least expenditure of energy.

To increase the degree of relaxation, muscle work is decreased, but it is not recoordinated for the purpose of changing skeletal relationships. Hence, relaxation would

* Jacobson, E. Theory of hypertension in man. Trans. N. Y. Acad. Sci. 11(2): 49–50. 1948.

tend to occur in accord with established patterns of neuro-muscular action, changing them but slightly, if at all. Whatever change may occur in muscle coordination will be undirected and in response to the need to maintain equilibrium and to move while trying to relax. Such automatic and undirected change may be for the better; or it may be for the worse, especially in certain postural deviations, such as lateral curvature of the spine or sacroiliac strain.

To increase the degree of muscular efficiency, in contrast to relaxation, there must be better alignment of the skeletal framework. This is attained only as muscle action is reconditioned to produce it. As the mechanics becomes better, the total result is less muscle work.

It seems logical then, to state that relaxation means less work in muscles. But it is not necessarily accompanied by an increased degree of efficiency, even though the metabolic rate may be lowered. On the other hand, an increased degree of efficiency means neuro-muscular recoordination, and it *is* accompanied by less muscle work.

To summarize, the development of the subject has dealt with: (1) the meaning of the terms, psychomotor, body mechanics, and posture; (2) the significance of conditioned reflexes in the self-direction of the human mechanism; and (3) how conditioned reflexes may be reconditioned toward the production of those mechanical considerations which promote decrease of strain in all human activities.

DIVISION OF MYCOLOGY

HISTOPLASMOSIS IN ANIMALS

C. W. EMMONS*

Much attention has been given to the special public health problems presented by bacterial diseases which affect both man and animals, but relatively little is known about this aspect of fungous diseases. Histoplasmosis, blastomycosis, sporotrichosis, coccidioidomycosis, and actinomycosis are some of the more important systemic mycoses which are common to man and animals. Accurate knowledge about their frequency and the possibility of their transmission from animals to man is fragmentary, although inspection of slaughtered animals in packing plants and scattered reports in medical and veterinary literature give some information about the occurrence in animals of the last two mentioned.[29] Coccidioidomycosis is known to occur also in rodents.[7]

Histoplasmosis in animals presents some unusual features, which deserve further consideration because of their intrinsic interest and because the study of histoplasmosis in animals promises to elucidate some of the puzzling problems in the epidemiology of the human disease. It should be pointed out at first that human and animal histoplasmosis have an identical etiology; both are caused by *Histoplasma capsulatum*. Strain differences are found among the isolates of the fungus, but these differences are not correlated with a particular host source. Fifteen strains recently isolated from animals in southern Georgia[12] were alike in one peculiar characteristic, which, however, was not seen in strains isolated from animals in Virginia but has been seen in a few human strains. This example serves to illustrate the significant point that *Histoplasma capsulatum*, like other fungi, varies or mutates freely. As increasing numbers of strains are available for study, it becomes apparent that, despite minor differences, one species, *H. capsulatum*, causes both human and animal histoplasmosis.

The study of histoplasmosis in animals is especially important at the present time, because of the current interest in human histoplasmosis. Proved histoplasmosis in man is a rare, almost invariably fatal disease, and only some 125 cases have been reported. There is a keen interest in the disease at the present time because the use of the histoplasmin skin test has shown that in some geographic areas as many as 80% of the general population react to this test.[3, 19, 21, 22]

* The Laboratory of Infectious Diseases, Microbiological Institute, National Institutes of Health, Bethesda, Maryland. This paper, illustrated with lantern slides, was presented before the meeting of the Division on April 22, 1949.

In some areas there is a high correlation between histoplasmin sensitivity and the occurrence of benign, nontuberculous, pulmonary calcification, which some investigators believe is a hitherto unrecognized form of histoplasmosis. Unfortunately, the histoplasmin skin test is not specific.[13] Clinical and laboratory confirmation in the diagnosis of a possible, very prevalent, benign histoplasmosis has not been obtained to date.

In the United States, the geographical areas of high histoplasmin sensitivity and of benign, nontuberculous, pulmonary calcification in humans coincide rather closely[3,21,22] and are limited, according to reported surveys, principally to a belt extending from eastern Kansas to the Atlantic coastal plain and from the middle of Iowa and northern Ohio southward through Tennessee. This sharp geographical limitation does not correspond to the known distribution of proved histoplasmosis. Although most of the reported cases have occurred in eastern United States, sporadic cases have occurred almost throughout the world, and it is not clear to what extent the distribution of the 125–150 human cases reflects either a true picture of the disease or medical interest in it and the utilization of laboratory facilities in its diagnosis.

A more precise knowledge of the actual distribution of histoplasmosis would aid in the interpretation of the equivocal histoplasmin skin test and in an understanding of the nature of benign, nontuberculous, pulmonary calcification. The detection of a sufficiently large number of human cases of proved histoplasmosis, even at the rate at which they have been reported during the past three or four years, does not offer an early answer to the questions raised. A systematic search for histoplasmosis in animals, especially in the domestic rat, does provide a practicable and conclusive method of demonstrating histoplasmosis in a given area. In our studies at the National Institutes of Health since November 1946, we have isolated *H. capsulatum* in culture from 48 naturally infected animals,[9, 10, 11, 12, 19, 20] and this probably equals the total number of strains of the fungus which have been isolated from human cases since DeMonbreun's classic studies 15 years ago. Application of the knowledge that the feral rat is susceptible to histoplasmosis and that naturally acquired histoplasmosis in the rat can be proved by the isolation of the fungus from the liver and spleen has already yielded important new information about the disease. Demonstration of histoplasmosis in rats or other animals provides the only practicable method now available for determining the geographical distribution of histoplasmosis.

I shall mention only briefly experimental histoplasmosis in laboratory animals. After DeMonbreun[5] isolated the fungus from a human case he reproduced the disease in monkeys and reported that puppies and mice were less susceptible. He subsequently[6] observed a natural infection in the dog and produced experimental infection in that animal. Hansmann and Schenken[14] reported experimental infection of the dog, rat, rabbit, and guinea pig. Parsons[24] described in considerable detail experimental histoplasmosis in mice. He observed generalized infection in mice when the mycelial form of the fungus was injected intravenously, but found only localized lesions when it was injected intraperitoneally. This has not been generally true in later investigations, and it may be that the use of a poorly sporulating strain was responsible for the result Parsons reported. The white mouse is a susceptible and satisfactory laboratory animal and develops a fatal infection when heavily inoculated with a virulent strain. When mice are given a small inoculum, they may develop a non-fatal form of the disease. This development of chronic histoplasmosis is notably evident, in my experience, in the rat and guinea pig. I have isolated *H. capsulatum* from guinea pigs two and one-half years after inoculation.[11] When killed, the animals appeared healthy and had no gross lesions at autopsy. By infecting a series of guinea pigs by intraperitoneal injection of a uniform dose of

conidia and killing the animals at suitable intervals, one can reconstruct the following sequence of events. The inoculum is quickly enveloped, and within 10–15 days a hard, tumor-like mass involving the omentum and pancreas develops. The original conidia and hyphal fragments of the inoculum can be found within abscesses in this mass. The liver and spleen become enlarged and the animal develops a systemic infection. Within a month, typically, the peritoneal lesions begin to regress, the liver and spleen return to normal size, and within a few weeks no gross evidence of infection remains; although *Histoplasma* can still be recovered in culture from the spleen and frequently also from the liver. The type of infection found in naturally infected wild animals appears to be similar to this chronic form developed in many experimentally infected animals.[10]

Historical. Naturally acquired histoplasmosis was unproved prior to 1947 in any animal except the dog. There are two early papers in which fungus infections resembling histoplasmosis were reported in mice and rats. The histopathological appearance was compatible with histoplasmosis, but the descriptions of the fungus were incomplete, notably as to cultures. Sangiorgi[26] described a rat captured in the laboratory, in which he found in the mononuclear cells of the enlarged spleen a fungus which he called a *Cryptococcus*. The description closely resembles that of the intracellular form of *Histoplasma*. It was reported that after 48 hours' incubation there developed in the cultures a *Cryptococcus*, which, when injected intraperitoneally, failed to produce disease in a guinea pig. No description of a mycelial type of growth was given.

Shortt[28] described, in 1 rat and 7 mice experimentally injected with flagellates, a fungus which, he stated, resembled Darling's *Histoplasma*, but which he named *Cryptococcus muri*. No cultures were described. More recently, Levine *et al.*[16] reported in a ferret a disease which may have been histoplasmosis, but cultures were not obtained. In the absence of data on cultures and in view of the writer's frequent isolation of a *Cryptococcus* from mice and rats,[9] some doubt exists regarding the actual nature of these fungi.

Dog. The dog was the first animal in which naturally acquired histoplasmosis was proved by adequate study and by the isolation of *H. capsulatum* in culture. The case, already mentioned, was reported very completely by DeMonbreun.[6] Thirteen cases of canine histoplasmosis have been reported since DeMonbreun's paper appeared,[1, 2, 19, 23, 27, 30, 31] and several additional unreported cases are known to be under study. The infected animals varied in age from 1 to 7 years. There were 7 males and 3 females among those whose sex was stated. Breeds represented were Boston bull, fox hound, Springer spaniel, Boston terrier, pekingese, mongrel, fox terrier, and English bull terrier. These animals came from Nashville, St. Louis, Des Moines, Brazil, Panama Canal Zone, Kansas, and northern Virginia. Tomlinson and Grocott[31] point out that their canine case from the Canal Zone represented the first demonstration of *Histoplasma* in that area since Darling's original 3 human cases 40 years before, although 15,000 autopsies had been done during the intervening period. Clinical and pathological findings were varied, but most of the dogs showed loss of appetite, emaciation, diarrhea, and enlargement of spleen, liver, and mesenteric nodes.

It is not possible to tell from the literature on canine histoplasmosis the probable source of infection. The cases reported have been sporadic and unrelated to each other, except for two litter mates reported by Harmon and those reported from northern Virginia, where two dogs from one litter and their sire had histoplasmosis, as proved by sections and cultures.[19, 27] Two other dogs from this litter died with undiagnosed diseases, and, although material was not available for study, it was believed that these dogs may have had histoplasmosis. The possibility that dogs may be infected from rats which they catch must be considered.

Mouse. Up to the present time, the isolation of Histoplasma from feral rodents has been reported only from our laboratory in the National Institutes of Health. Since November 1945, a small scale but persistent and more or less systematic attempt has been made to gain additional knowledge about histoplasmosis by a search for animal hosts of the disease. This search has been conducted in Virginia, Maryland, District of Columbia, Iowa, and Georgia. Attention has been directed at one time or another toward several species of mice, including the meadow vole (*Microtus spp.*), white footed mouse (*Peromyscus spp.*), shrew (*Blarina brevicaudata*), and pine mouse (*Pitymys pinetorum*). The reasons for selecting these species for study have not as yet been justified by results. However, among more than 1000 house mice (*Mus musculus*) trapped, one animal with proved histoplasmosis was found.[19] This was trapped at a house where one of the infected dogs mentioned above had lived more than a year before. No gross lesions were observed when this mouse was killed. Histologic sections showed only microscopic epithelioid granulomata in lung, liver and spleen, and a few fungus cells.

Rat. The search for histoplasmosis in the rat has been more successful in revealing an animal reservoir of histoplasmosis. Twenty-three brown rats (*Rattus norvegicus*) with proved histoplasmosis, representing slightly more than 2% of those trapped, have been found in Loudoun County, Virginia.[9, 10, 11] These infected animals have been captured on several widely separated farms in this rural county. No importance is attached to this percentage, since no attempts were made to take a random sample. When infected rats were once found on a farm premise, trapping was continued at that location in an attempt to learn more about the local occurrence of histoplasmosis.

Rats were captured in steel traps, brought to the laboratory alive when possible, and the autopsy was performed promptly after the animal was killed. Sterility precautions were observed in the autopsy, since the presence of bacterial or fungus contaminants frequently inhibit growth of Histoplasma. Cultures were made from liver, spleen, adrenal, urinary bladder, and lung by streaking a piece of tissue on a Sabouraud agar slant. The cultures were incubated at 30°C for 3 or 4 weeks before they were discarded as negative.

No correlation between gross lesions observed at autopsy and the isolation of *Histoplasma* was observed. In most of the infected rats the spleen and liver were normal in size and appearance. Histopathologic studies of these and other organs involved showed only microscopic, epithelioid cell granulomata.[10] *Histoplasma* was found in small numbers in some infected rats and not at all in a few, although microscopic lesions were present. So far as could be determined from the infected animals examined, histoplasmosis is a benign infection in the brown rat. This is in agreement with observations of histoplasmosis in the laboratory strain of white rat used in experimental studies of histoplasmosis.[9]

The search for histoplasmosis in the rat was extended to south Georgia where, in collaboration with the Typhus Investigations unit of the Communicable Disease Center, U.S.P.H.S., some 1300 rats have been cultured.[12] These studies resulted in the isolation of *Histoplasma* from 7 brown rats and 4 roof rats (*R. rattus*). The strains of *H. capsulatum* isolated from Georgia rats differ in one respect from those isolated in Virginia. They produce enormous numbers of microconidia and relatively few macroconidia. In this respect they resemble a few strains isolated from man, notably those described by Negroni[18] from South American cases. Further study may reveal important geographic differences in strains of *Histoplasma*, but no adequate reason appears at the present time for splitting the species *H. capsulatum* on the basis of differences observed among these rodent strains.

Cat. The demonstration of histoplasmosis in the rat suggested at once an examination of the cat, since many farm cats are rat-catchers. During the past two years,

we have killed and made cultures from 49 cats collected in Loudoun County, Virginia. Five of these had histoplasmosis, as proved by culture.[19] The first infected cat was obtained in July, 1948. Three of those with histoplasmosis came from a farm where 8 of 124 rats previously trapped had this disease. Thus, it seems probable that the cat is infected by eating rats with histoplasmosis.

A surprising feature of histoplasmosis in the cat is the limited extent of the lesions. Histoplasma was isolated from the spleen in only one animal. In the other four the only isolation was from one or more of the cervical lymph nodes.

There is one previous report of histoplasmosis in the cat, but, as Meleney[17] has pointed out, this was an erroneous diagnosis for toxoplasmosis.

Skunk. The highest incidence of infected animals was found in the spotted skunk (*Spilogale putorius*). Five of six skunks trapped and cultured in southern Georgia were infected.[12] Isolations were from spleen and liver in these animals. The skunk is a carnivore and is known to eat rats. A further study of this animal and of other related species is indicated.

Horse. There is one report of histoplasmosis in the horse.[25] The diagnosis was made entirely by identification of the fungus in sections of lung tissue. Since the fungus causing epizootic lymphangitis in horses is indistinguishable from *H. capsulatum* in tissue, some doubt might be entertained concerning the correctness of the diagnosis.

In reviewing the status of proved histoplasmosis in animals, the dog may be listed first because of the chronological order of reports, the recognition of canine histoplasmosis independently by several investigators, and the intimate association between man and the dog. Our studies have first proved the occurrence of histoplasmosis in *Mus musculus*,[19] *Rattus norvegicus*,[11] *R. rattus*,[12] *Felis domestica*,[20] and *Spilogale putorius*.[12] In all except the house mouse, *Histoplasma capsulatum* has been isolated from substantial numbers of individuals. All the animals, with the exception of the spotted skunk, come into rather intimate contact with man, his food and clothing, or other objects in his environment. Whether the exposure of man to animals with histoplasmosis is important in the epidemiology of human histoplasmosis is a question of considerable importance. Unfortunately, we are unable to answer it at the present time. The limited extent of infection in most of the animals studied[10] suggests the improbability of direct transmission from animals to man either by ectoparasites or exposure to excreta.

One item of collateral evidence may be pertinent. The demonstration of histoplasmosis in rats stimulated a search for a free-living or saprophytic form of *Histoplasma* in the environment of infected animals. During the past two years, 387 samples of soil or various types of debris have been collected on farms from which infected animals have been collected. By suspending soil in physiological saline and injecting the upper portion of such a suspension into mice, *H. capsulatum* has been isolated from two soil samples.[8]

In addition to the isolation of *Histoplasma* from mice infected from these two soil samples, the typical macroconidia of *H. capsulatum* were visibly demonstrated by microscopic examination of one of these suspensions.[8] Since these macroconidia have never been found developing in the animal body but are known to develop freely in the saprophytic growth cycle of the fungus, their demonstration in soil seems to indicate that there is a free-living form of *Histoplasma* in the environment of man and animals. Therefore, it is not necessary to postulate transmission of histoplasmosis directly from man to man or animal to man in order to explain the occurrence of human histoplasmosis. The possibility exists, however, that the seeding of soil by the eventual death of animals with chronic histoplasmosis is important in maintaining *Histoplasma* in the soil flora.

The significance of an animal reservoir of

histoplasmosis is still obscure. The fact that such cosmopolitan animals as the rat, cat, and dog are important hosts of *Histoplasma* may be significant in the world-wide distribution of human histoplasmosis. A comparison with coccidioidomycosis is interesting. Coccidioidomycosis also occurs in the dog, but, among the native wild animals present in the limited endemic area of coccidioidomycosis, only species of *Perognathus*, *Dipodomys*, and *Citellus* peculiar to the desert have so far been shown to be important hosts of *Coccidioides*. A possible necessary relationship to these desert-dwelling rodents is the only explanation which has so far been offered with any supporting evidence to account for the limited geographical distribution of coccidioidomycosis.[7]

To summarize, naturally occurring histoplasmosis has been proved in the dog, brown rat, roof rat, domestic cat, and the spotted skunk by isolation of *H. capsulatum* in culture. The detection of histoplasmosis in these or other animals is the only practicable method now available for determining conclusively the geographic distribution of histoplasmosis.

REFERENCES

1. Anthony, C. H. 1948. Case history—histoplasmosis (canine). Jen-Sal Jour. Nov.–Dec: 22.
2. Birge, R. F. & W. H. Riser. 1945. Canine histoplasmosis. Report of two cases. N. Amer. Vet. **26:** 281–287.
3. Christie, A. & J. C. Peterson. 1945. Pulmonary calcification in negative reactors to tuberculin. Am. J. Pub. Health. **35:** 1131–1147.
4. Callahan, Jr., W. P. 1944. Spontaneous histoplasmosis occurring in a dog. Am J. Trop. Med. **24:** 363–366.
5. DeMonbreun, W. A. 1934. The cultivation and cultural characteristics of Darling's *Histoplasma capsulatum*. Am. J. Trop. Med. **14:** 93–125.
6. Idem. 1939. The dog as a natural host for *Histoplasma capsulatum*. Report of a case of histoplasmosis in this animal. Am. J. Trop. Med. **19:** 565–587.
7. Emmons, C. W. 1943. Coccidioidomycosis in wild rodents. A method of determining the extent of endemic areas. Pub. Health Repts. **58:** 1–5.
8. Idem. The isolation of Histoplasma from soil. In press.
9. Idem. Unreported data.
10. Emmons, C. W. & L. L. Ashburn. 1948. Histoplasmosis in wild rats. Occurrence and histopathology. Pub. Health Repts. **63:** 1416–1422.
11. Emmons, C. W., J. A. Bell & B. J. Olson. 1947. Naturally occurring histoplasmosis in *Mus musculus* and *Rattus norvegicus*. Pub. Health Repts. **62:** 1642–1646.
12. Emmons, C. W., H. B. Morlan & E. Hill. The occurrence in Georgia of histoplasmosis in rats (*Rattus norvegicus* and *R. rattus*) and skunks (*Spilogale putorius*). In press.
13. Emmons, C. W., B. J. Olson & W. W. Eldridge. 1945. Studies of the role of fungi in pulmonary disease. I. Cross reactions of histoplasmin. Pub. Health Repts. **47:** 1383–1394.
14. Hansmann, G. H. & J. R. Schenken. 1934. A unique infection in man caused by a new yeast-like organism, a pathogenic member of the genus Sepedonium. Am. J. Path. **10:** 731–738.
15. Harmon, K. S. Histoplasmosis in dogs. 1948. A report of two cases. J. Amer. Vet. Med. Assoc. **113:** 60–62.
16. Levine, N. D., G. L. Dunlap & R. Graham. 1938. An intracellular parasite encountered in ferret. Cornell Veterinarian. **28:** 249–251.
17. Meleney, H. E. 1945. Toxoplasmosis mistaken for histoplasmosis in a cat. Amer. J. Trop. Med. **25:** 163.
18. Negroni, P. 1940. Estudio micologico del primer caso sud-americano de histoplasmosis. Rev. Insto. Bacto. **9:** 239–294.
19. Olson, B. J., J. A. Bell & C. W. Emmons. 1947. Studies on histoplasmosis in a rural county. Am. J. Pub. Health. **37:** 441–449.
20. Olson, B. J., C. W. Emmons & L. L. Ashburn. The occurrence of Histoplasmosis in the cat. In press.
21. Palmer, C. E. 1945. Nontuberculous pulmonary calcification and sensitivity to histoplasmin. Pub. Health Repts. **60:** 513–520.
22. Idem. 1946. Geographic differences in sensitivity to histoplasmin among student nurses. Pub. Health Repts. **61:** 475–487.
23. Para, M. 1946. Histoplasmosis in Brazil Amer. J. Trop. Med. **26:** 273–292.
24. Parsons, R. J. 1942. Experimental histoplasmosis in mice. Arch. Path. **34:** 229–239.
25. Richman, H. 1948. Histoplasmosis in a colt. N. Amer. Veter. **29:** 710.
26. Sangiorgi, G. 1922. Blastomicosi spontanea nei Muridi. Pathologica. **14:** 493–495.
27. Seibold, H. R. 1946. A case of histoplasmosis in a dog. J. Amer. Vet. Med Assoc. **59:** 209–211.
28. Shortt, H. E. 1923. The pathogenicity of insect flagellates to vertebrates, with special reference to *Herpetemonas ctenocephali* Fantham. Indian J. Med. Res. **10:** 908–933.
29. Stiles, G. W. & C. L. Davis. 1942. Coccidioidal granuloma (Coccidioidomycosis). Its incidence in man and animals and its diagnosis in animals. J. Amer. Med. Assoc. **119:** 765–769.

30 THURINGER, J. M. 1944. Histoplasmosis. Report of its occurrence in a dog. Arch. Path. **37**: 140–142.
31. TOMLINSON, W. J. & R. G. GROCOTT. 1945. Canine histoplasmosis. A pathologic study of the three reported cases and the first case found in the Canal Zone. Am. J. Clin. Path. **15**: 501–507.

SECTION OF ANTHROPOLOGY

EVIDENCE FOR PLEISTOCENE MAN AT LA JOLLA, CALIFORNIA

By GEORGE F. CARTER*

Introduction. Exposed in the seaward face of an alluvial fan now undergoing active destruction through wave attack is evidence that man lived at La Jolla, California, throughout most, if not all, of the period when this fan was being built. This fan is so situated that it is a sensitive recorder of land and sea level relations. It is not only capped by a mature soil, but it contains buried soil profiles from which some temporal significance can be gleaned. From a combined morphological and pedological study of this fan, some insight into the antiquity of man in Southern California can be gained.

The evidence for man consists of hearths, the shells from seafoods, stone flakes produced by man, and one stone used for the grinding of seeds (technically known as a mano). This material is distributed as follows: hearths, flakes, and shell are to be found throughout the 20-foot thick exposed depth of the fan; the mano was found at a depth of five feet. Careful study of the fan has shown that the depth of burial is not particularly significant, since the fan has been subjected to past periods of erosion and deposition, and some of the deeper materials are younger than some of the higher materials.

Morphology of the Scripps Fan. This fan is now being destroyed by the sea, and its truncated face forms a low cliff 20 to 30 feet high. It is probably significant that the fan has been under rapid retreat in the present period of rapidly rising sea level. At any rate, the fact that the alluvial fan is being *destroyed* under the present conditions indicates that it could not have been *built* under the existing conditions. Both the submarine contours off this beach and a projection of the remnant of the fan indicate that when it was built it must have extended approximately one half mile seaward of its present terminus. The depth of water indicated at the probable outer edge of the fan would then be at 100 feet. This may be taken as an approximation of the minimum amount of lowering of the sea in relation to the land to allow the fan to grow.

The evidence for the origin of the fan may then be stated as follows: At a time when the sea stood in relation to the land 100 feet or more lower than at the present, a gently sloping platform approximately three quarters of a mile wide (one half mile now under sea, plus the one quarter mile still above sea level) was exposed at the foot of the 300 cliff terminating the seaward side of the Linda Vista Mesa. Alluvium eroded from a portion of this mesa was deposited where a small canyon opened onto this shelf, and a sharp reduction of gradient occurred. Under these conditions, an alluvial fan was constructed. With a subsequent rise of sea level in relation to the land, the unconsolidated alluvium has been exposed to wave attack and has retreated approximately one half mile.

Dissection of the Fan. The fan is now cut by a deep gully. This has resulted both from the cliffing due to truncation and the associated steepening of the gradient of the fan. The face of the cliff reveals marked horizontal bands of differing sedimentary materials. Close study reveals that these

* The Johns Hopkins University, Baltimore, Maryland. This paper, illustrated with lantern slides, was presented before the meeting of the Section on April 25, 1949.

bands are not continuous but that in the past they have been cut by gullies comparable to the existing one. These old gullies, however, had been filled and the fan resumed its growth. Careful mapping of the gullies and the principal strata reveals that there have been three major periods of alluviation and of gully-cutting. In terms of the mechanics of accumulation versus truncation and gully-cutting, this sequence of events would seem to call for three cycles of low sea (or high land) level followed by high sea (or low land) level.

Fire removal of the protective cover of the fan and its catchment source, extreme floods, and minor climatic fluctuations cannot be arbitrarily ruled out as possible causes of the sequence of gullying and erosions that occurred on this alluvial fan. All of them are theoretically possible causes. On the other hand, the mechanism of fluctuating land or sea level would not only theoretically produce the result, but, under conditions of present rising sea levels, *is* producing results comparable to what has occurred in the past. Lacking any other clear mechanism to produce the observed gullying and filling, and possessing evidence that the present gullying is correlated with a rising water level, it seems logical to incline to the theory of gullying and alluviating as resulting from alternating land-sea level relationships.

Eustatically Fluctuating Sea Level and the La Jolla Fan. The question of land or sea stability is capable, in part, of simple solution. The sea level is known to have changed during the Pleistocene with the addition and subtraction of water due to the melt or accumulation of ice. The sea level is now rising due to the addition of melt water. Sea level during a glacial period can never have been fixed, for the glaciers are known to have waxed and waned within each glacial period. Since we are in the terminal part of a glacial period, the sea level cannot be stable now, nor can it have been in the glacial past. The answer to our question of sea or land movement or both must then be: the sea level has surely been fluctuating; the land level may have been. The movement of sea level can be assumed. Changes of land level must be proven. The evidence from this immediate vicinity suggests a relatively stable coast. Data from regions to the north are conflicting. The Ventura and Los Angeles areas have been described as zones of active crustal movement. A recent study of the Santa Barbara region, however, indicates conditions of stable land and fluctuating sea that are closely comparable to the situation in San Diego county.

In light of the evidence now at hand, the following conclusions seem to be indicated. The low elevation of this fan in an area marked by 25, 60, and 100 foot beaches and terraces indicates that it is younger than these sea stands. The evidence for relative crustal stability in this area throughout at least the upper Pleistocene and the absence of proof for any other mechanism creating this pattern of alternating gullying and filling, taken with the known sea level fluctuation, all combine to suggest that the causal factor in the cycle of gullying and filling may well have been a fluctuating sea level against a relatively stable land mass. If such is the case, the time of the building of this fan must have been during the last eustatically lowered sea level. In terms of glacial chronology, this means the fan grew during Wisconsin times.

Internal Structure and Time. None of the erosions were so extensive as to remove all of the original fan surface. This is evidenced by the ease with which old surfaces can be traced through the length of the face of the fan. These periods of erosion are considered to represent periods of relatively high sea stand and probably to have been the result of truncation and gullying comparable to the present. During each such period of gullying, strongly developed soil profiles developed on the fan surface. The one capping the present surface is described in the soil survey of the region as a mature soil. This implies an age of many thousands of years. The buried soil beneath this is approximately twice as

strongly developed. A third such soil profile is associated with the earliest of the major erosions.

If we possessed a reliable figure for post glacial time, we would now be in a position to make some rough computations as to the age of the strata related to these soil horizons. Antevs has estimated that the sea reached within 100 feet of modern levels about 10,000 years ago. Truncation of the fan, with the consequent formation of the modern gully, should date from that time.

An attempt at a time estimate, based on 10,000 years as the assumed onset of truncation, gullying, and initiation of soil formation, is given in TABLE 1. Note that no time has been allowed for gully-cutting, although this process was certainly not instantaneous. Only 5,000 years have been

TABLE 1

Geomorphic Cycle	*Suggested Dates*
Present erosion and soil weathering	Began 10,000
Deposition	5,000
Erosion and development of first buried soil	20,000
Deposition of materials, including mano	5,000
Estimated age of mano	40,000 years

allowed for the filling of the gullies and the deposition of a new layer of material over the fan surface. An allowance of 5,000 years is probably minimal. Consider that the foot of the fan had to be completely rebuilt before the gully could fill and the entire fan surface alluviate. Further, the deposition on this fan, whose surface is large in proportion to its drainage basin, was accomplished by an intermittent stream, and the materials in the fan indicate gentle deposition rather than thick flood deposits. The figure of 40,000 years ago for the beginning of the development of the buried soil profile may, therefore, be a minimum figure. An attempt to date materials associated with this horizon is of interest, since it is in this horizon that evidence for man consists of such positive materials as food, shell, stone flakes, and one mano.

The evidence so far, then, suggests that man, possessing the mano and by inference using the metate to grind wild seeds, eating shell fish, and possessed, so far as is now known, of only simple stone flakes, lived at La Jolla something like 40,000 years ago. If the hearths found in deeper strata are indeed evidence of man, then human occupancy of this area extends back through the Wisconsin period. One piece of evidence exists, to be reported elsewhere, for associating man with a 25 foot beach level here. If this coast is proven relatively stable, this would indicate interglacial man and probably double the antiquity suggested above. The poverty of the materials in the alluvial fan do not suggest a people with any traces of a culture suitable to glacial arctic climate survival. This suggests that bearers of this culture entered America via Bering Straits during some milder climatic period.

It should be noted that it is not possible to extrapolate from comparative culture levels to comparable dates elsewhere in America. California, and especially southern and lower California, are regions of extreme cultural lags. Such primitive cultures (in terms of imperishable materials) as we are here dealing with *might* have been contemporaneous with more developed lithic cultures elsewhere in California or America. At this early period, however, such cultural differentiation in the Americas seems to me unlikely.

There seems to be considerable validity to the sequence of lithic industries in this area and their indicated relative antiquity. Despite considerable effort, I have yet to find a developed tool, except for the mano reported here, beneath a comparably developed soil profile. It can be argued that we have simply failed to find a full assemblage of implements. This is possible, but the fact that the site has been under observation for nearly twenty years makes it improbable. Further, other sites in San Diego county beneath similar soil profiles also fail to produce developed implements. Beneath such soils as cap this site, I have yet to find more than unretouched flakes.

A final extension of these findings has considerable interest. If man, of as simple a culture as has here been described, was living on the coast of California at a time when large masses of ice still existed in the northern part of the continent, it raises the question of how he entered America. There is nothing to suggest that he had a boat culture that would enable him to cross the oceans. He must, then, have come by a land bridge. The Bering straits offers almost the only possibility. Since the evidence at La Jolla seems related to lowered sea levels and this to continental ice sheets, the Bering region must have been colder than at present. Any ice free corridor such as has been suggested for the interior of the continent, if only due to the refrigerating effect of the ice masses, must have been greatly colder than the coastal sections of the Bering region. Yet even today, when the arctic is warmer than it could have been during the period of continental ice sheets, it requires a technologically highly developed culture to survive in the Bering region. The evidence at La Jolla, and in San Diego county generally, indicates that technologically advanced cultures are very much later than the material in the site here described.

One might, of course, argue that the La Jolla people are decadent survivals of higher culture. There are, however, no known earlier cultures from which to derive this culture. If there were, even more time would be needed to account for such a change. To explain the phenomena of so primitive a culture in America at so early a time, it seems necessary to assume that its progenitor passed through the Bering strait region at a time when the climate was markedly warmer than at present and when the great continental ice masses did not exist. This is equivalent to suggesting that man entered America during the last interglacial period.

In the past, it was frequently pointed out that the absence of pre-*sapiens* man in America was evidence of the *Recent* (non-*Pleistocene*) time of entry of man into America. Our new knowledge of the antiquity of *sapiens* man invalidates this argument. Such sites as I have reported this evening, together with numerous other reports of primitive cultures suggesting great antiquity for man in America, lead to the conclusion of the probability of man entering America in the last interglacial period.

CONFERENCE HELD

SECTION OF BIOLOGY

"INTERNATIONAL CONFERENCE ON VITAMIN E"

APRIL 14 AND 15, 1949

The Section of Biology held an "International Conference on Vitamin E." Doctor Karl E. Mason, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y., was the Conference Chairman in charge of the meeting.

The program consisted of the following papers:

Friday, April 15

MORPHOLOGIC LESIONS IN VITAMIN E DEFICIENCY

Morning Session. Chairman, A. M. Pappenheimer, Department of Bacteriology and Immunology, Harvard Medical School, Boston, Mass.

Opening of the Conference, Karl E. Mason.

Introductory Remarks by the Session Chairman, A. M. Pappenheimer.

"Effect of Ovarian Hormones upon

Uterine Pigmentation in Vitamin E-Deficient Rats," by W. B. Atkinson, H. Kaunitz and C. A. Slanetz, Departments of Anatomy, Pathology, and Animal Care, College of Physicians and Surgeons, Columbia University, New York, N. Y.

"Histochemistry of Uterine Pigment in Vitamin E-Deficient Rats," by Herbert Elftman, H. Kaunitz and C. A. Slanetz, Departments of Anatomy, Pathology, and Animal Care, College of Physicians and Surgeons, Columbia University, New York, N. Y.

"Restoration of the Vaginal Estrus by Alpha-Tocopherol Acetate in Old Rats," by S. Y. P'an, H. Kaunitz and C. A. Slanetz, Departments of Pharmacology, Pathology, and Animal Care, College of Physicians and Surgeons, Columbia University, New York, N. Y.

"Increased Tocopherol Requirements During the Rat's Menopause," by H. Kaunitz and C. A. Slanetz, Departments of Pathology and Animal Care, College of Physicians and Surgeons, Columbia University, New York, N. Y.

"On the Biological Assay of Vitamin E by the 'Male Rat Test,'" by Maria Luisa Herraiz and Juan Carlos Radice, National Institute of Nutrition, Buenos Aires, Argentina.

"Vitamin E Studies on Mice with Special Reference to the Distribution and Metabolism of Lipids," by Z. Menschik, M. K. Munk, T. Rogalski, O. Rymaszewski and T. J. Szczesniak, Departments of Anatomy, Polish School of Medicine, Edinburgh, and School of Medicine, University of Ottawa, Ottawa, Canada.

"Ten Year Incidence of Field Encephalomalacia in Chicks and Observations on Its Pathology," by Erwin L. Jungherr, Department of Animal Diseases, Storrs Agricultural Experiment Station, University of Connecticut, Storrs, Conn.

"Central Nervous System Changes in Chronic Vitamin E-Deficient Rats," by Charles N. Luttrell and Karl E. Mason, Department of Anatomy, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.

Papers Read by Title

"Prolonged Avitaminosis E in Rats and Fibrosis of the Endometrium," by J. Lopes de Faria and J. Dutra de Oliveira, Departments of Pathology and Physiology, Faculty of Medicine, University of Sao Paulo, Sao Paulo, Brazil.

"Ceroid Substance and Its Meaning," by J. Dutra de Oliveira, Department of Physiology, Faculty of Medicine, University of Sao Paulo, Sao Paulo, Brazil.

"The Flourescent Pigment in the Uteri of Rats in Avitaminosis E," by J. C. Radice and Maria Luisa Herraiz, National Institute of Nutrition, Buenos Aires, Argentina.

"The Fluorescent Pigment in Atrophic Testes of Rats in Avitaminosis E," by Enrique Pierangeli, J. C. Radice and Maria Luisa Herraiz, National Institute of Nutrition, Buenos Aires, Argentina.

"The Effects of Hypo- and Hyper-vitaminosis E on Lung Tumor Growth in Mice," by I. R. Telford, Department of Anatomy, School of Dentistry, University of Texas, Houston, Texas.

"The Effect of Chronic Vitamin E Deficiency on the Nervous System of the Rat," by M. Malamud, Marjorie M. Nelson and Herbert M. Evans, Laboratory of the Langley Porter Clinic, San Francisco, and the Institute of Experimental Biology, University of California, Berkeley, Calif.

"Studies on the Histopathology of Vitamin E Deficiency," by Raoul Lecoq and Pierre Isidor, Hospital de Saint-Germain-en-Laye, Paris, France.

"Action of Vitamin E on the Non-Deficient Organism," by Raoul Lecoq, Paul Chauchard and Henriette Mazque, Hospital de Saint-Germain-en-Laye, Paris, France.

"Influence of Tocopherols, Olive Oil, Testosterone and Progesterone on Avitaminosis E Lesions," by Enrique Pierangeli, Domingo Mosto, Maria Luisa Herraiz, Juan Carlos Radice and Francisco J. Cortada, National Institute of Nutrition, Buenos Aires, Argentina.

TOCOPHEROLS AND THEIR ESTERS IN ENZYME AND TISSUE FUNCTIONS

Afternoon Session. Chairman, H. A. Mattill, Department of Biochemistry, College of Medicine, State University of Iowa, Iowa City, Iowa.

Introductory Remarks by the Session Chairman, H. A. Mattill.

"Techniques for Studying the Tocopherols and Their Phosphates in Enzyme Systems," by Stanley R. Ames, Research Laboratories, Distillation Products, Inc., Rochester, N. Y.

"Some Chemical and Enzymic Alterations in Muscles in Experimental Dystrophy," by Charlotte E. Roderuck, Daniel H. Basinski and Mary Alice Barber, Department of Biochemistry, College of Medicine, State University of Iowa, Iowa City, Iowa.

"Alpha - Tocopherol, Alpha - tocopheryl Phosphate, and Phosphorylation Mechanisms," by William M. Govier and Anna J. Gibbons, Department of Pharmacology and Endocrinology, The Upjohn Company, Kalamazoo, Mich.

"The Inhibition of Hyaluronidase by Alpha-Tocopherol Esters," by Wilbur H. Miller and Alice M. Dessert, Chemotherapy Division, Stamford Research Laboratories, American Cyanamid Company, Stamford, Conn.

"On the Effects of Alpha-tocopheryl Phosphate on Oxidative Mechanisms in Striated Muscle," by K. L. Zierler, B. P. Folk, D. Grob, C. Eyzaguirre, L. W. Jarcho and J. L. Lilienthal, Jr., Department of Medicine, The Johns Hopkins Hospital, Baltimore, Maryland.

"Chemical and Biological Studies Related to the Metabolic Function of Vitamin E," by Paul D. Boyer, Division of Biochemistry, University of Minnesota, St. Paul, Minn.

"Relationship of Vitamin E Deficiency to Tissue Peroxides," by Henrik Dam, Department of Biology, Polytechnic Institute, Copenhagen, Denmark.

PROTECTIVE ACTIONS OF VITAMIN E IN CONDITIONS OF METABOLIC STRESS

Evening Session. Chairman, Cosmo G. Mackenzie, Department of Biochemistry, Cornell University Medical College, New York, N. Y.

Introductory Remarks by the Session Chairman, Cosmo G. Mackenzie.

"The Significance of Protein in Vitamin E Deficiency," by Thomas Moore, Dunn Nutritional Laboratory, Cambridge, England.

"Comparison of a Fatal Tocopherol-Deficiency Vascular Disease in Rats with the Syndrome Caused by CCl_4 Toxicity," by Edwin L. Hove, Division of Nutrition, Alabama Polytechnic Institute, Auburn, Alabama.

"Dietetic Hepatic Injuries and the Mode of Action of Tocopherol," by Klaus Schwarz, Johannes-Gutenberg University, Mainz, Germany.

"Tocopherol and Hemolysis *in Vivo* and *in Vitro*," by Paul Gyorgy and Catharine S. Rose, Nutritional Service of the Department of Pediatrics, and the Gastro-Intestinal Section of the Medical Clinic, School of Medicine, University of Pennsylvania, Philadelphia, Penna.

Saturday, April 16

PRACTICAL NUTRITIONAL ASPECTS OF VITAMIN E

Morning Session. Chairman, P. L. Harris, Research Laboratories, Distillation Products, Inc., Rochester, N. Y.

Introductory Remarks by the Session Chairman, P. L. Harris.

"The Relation of Vitamin E to Reproduction in Dairy Cattle," by T. W. Gullickson, Department of Dairy Husbandry, University of Minnesota, Minneapolis, Minn.

"Effects of Tocopherols in Vitality of Pigs in Relation to 'Baby Pig Disease,'" by L. E. Carpenter and W. O. Lundberg, University of Minnesota, Minneapolis, Minn., and The Hormel Institute, Austin, Minn.

"Vitamin E Requirement and Economy

in Farm Animals," by J. K. Loosli, Laboratory of Animal Nutrition, Cornell University Agricultural Experiment Station, Ithaca, N. Y.

"Studies on Vitamin E Deficiency in the Monkey," by L. J. Filer, Jr., Ruth E. Rumery, Nan Gan Yu and Karl E. Mason, Departments of Anatomy and Medicine, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.

"Tocopherols in Food and Tissues," by Mary L. Quaife, Research Laboratories, Distillation Products, Inc., Rochester, N. Y.

"Vitamin E in Human Nutrition," by Chr. Engel, Central Institute for Nutrition Research T. N. O., Utrecht, The Netherlands.

"Serum Vitam E Levels in Complications of Pregnancy," by Nevin S. Scrimshaw, Roy B. Greer and Ruth L. Goodland, Department of Obstetrics and Gynecology, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.

"Plasma Tocopherols in Health and Disease," by William J. Darby, Mary Ellen Cherrington, Robert H. Furman, Janet Lemley and George R. Meneely, Division of Nutrition, Departments of Medicine and Biochemistry, Vanderbilt University School of Medicine, Nashville, Tenn.

Papers Read by Title

"On the Chromatographic Separation of Tocopherols," by A. Emmerie, The Laboratory of Hygiene of the University, Utrecht, The Netherlands.

"Vitamin E in the Nutrition of Farm Animals," by D. B. Parrish, Kansas Agricultural Experiment Station, Manhattan, Kansas.

"Response of Swine to Vitamin E Deficient Rations," by F. B. Adamstone, J. L. Krider and M. F. James, Departments of Zoology and Animal Science, University of Illinois, Urbana, Ill.

"The Vitamin E Blood Level in Cows and Horses under Different Conditions," by F. C. van der Kaay, Veterinary Gynecological Institute of The Netherlands, State University, Utrecht, The Netherlands.

"The Vitamin E Content of Edible Oils Consumed in Buenos Aires (Argentina)," by Enrique Periangeli, Maria Luisa Herraiz and Herminia G. de Alvarez Herrero, National Institute of Nutrition, Buenos Aires, Argentina.

"Shute Test for Checking Unbalance Produced by Lack of Vitamin E," by J. Dutra de Oliveira, Medical School, University of Sao Paulo, Sao Paulo, Brazil.

"The Significance of Serum-Tocopherol Levels during Pregnancy," by Lauri Rauramo, Women's Clinic, University of Helsinki, Helsinki, Finland.

"Experimental and Clinical Studies on Metabolic Interrelationships of Vitamin E," by Ugo Butturini, Institute of Medical Pathology, University of Parma, Parma, Italy.

VITAMIN E IN CLINICAL MEDICINE

Afternoon Session. Chairman, R. S. Goodhart, Scientific Director, National Vitamin Foundation, Inc., New York, N. Y.

Introductory Remarks by the Session Chairman, R. S. Goodhart.

"Comparison of the Effects of Alpha-Tocopherol and Matching Placebo on Chest Pain in Patients with Heart Disease," by J. Travell, S. H. Rinzler, H. Bakst, Z. H. Benjamin and A. L. Bobb, Cardiovascular Research Unit of Beth Israel Hospital, and Department of Pharmacology, Cornell University Medical College, New York, N. Y.

"Negative Results of Tocopherol Therapy in Cardiovascular Disease," by Charles K. Donegan, Addison L. Messer, E. S. Orgain and J. Ruffin, Department of Medicine, Duke University School of Medicine, Durham, North Carolina.

"Vitamin E in Cardiovascular Disease, with Special Reference to Vascular Occlusion and Hypertension," by Evan V. Shute and Wilfred E. Shute, Department of Medicine, The Shute Institute for Clinical and Laboratory Medicine, London, Ontario, Canada.

"Tocopherol Therapy in Stasis Ulcer and Stasis Dermatitis," by Conrad Stritzler, Dermatological Services, Queens General

Hospital and Long Island College Hospital, Brooklyn, N. Y.

"Vitamin E in Rheumatic Disease," by Morris Ant and Erwin Di Cyan, Brooklyn, N. Y., and Di Cyan and Brown, New York, N. Y.

"Vitamin E and Collagen in the Rheumatic Diseases," by C. L. Steinberg, Rochester General Hospital, Rochester, N. Y.

"Tocopherols in Peyronies' Disease (A Follow Up)," by William Wallace Scott and Peter L. Scardino, The James Buchanan Brady Urological Institute, The Johns Hopkins Hospital, Baltimore, Maryland.

"Vitamin E in the Treatment of Diabetes Mellitus," by Arthur Vogelsang, London, Ontario, Canada.

Papers Read by Title

"Failure of Vitamin E to Increase Sperm Counts in Men," by Edmund J. Farris, Wistar Institute of Anatomy and Biology, Philadelphia, Penna.

"A Trial of Vitamin E Therapy in Juvenile Diabetes Mellitus," by George M. Guest, Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, Ohio.

"Some Experiences with the Use of Vitamin E in Various Cardiac Conditions," by Samuel Baer, William I. Heine and D. Barton Gelfond, Cardiac Department, Jewish Hospital, Philadelphia, Penna.

"Tocopherol Therapy in Buerger's Diseased and Varicose Ulcers," by L. L. Pennoch, Pittsburgh, Penna.

"Alpha-Tocopherol in Alleviating Chorea," by G. C. Dowd, Boston Evening Clinic and Hospital, Boston, Mass.

"Massive Doses of Alpha-Tocopherol in Multiple Sclerosis," by G. C. Dowd, Boston Evening Clinic and Hospital, Boston, Mass.

"Tocopherols in Urethral Strictures," by Peter L. Scardino and Perry B. Hudson, The James Buchanan Brady Urological Institute, The Johns Hopkins Hospital, Baltimore, Maryland.

NEW MEMBERS

Elected April 28, 1949

LIFE MEMBERSHIP

Raymond, Albert L., Ph.D., Biochemistry. Vice-President in Charge of Research and Development, G. D. Searle & Co., Chicago, Illinois.

SUSTAINING MEMBERSHIP

Burket, Louis C., M.D., Pediatric Physiology. Associate Pediatrician, Mercy Hospital, Altoona, Pa.

Jarcho, Julius, M.D., Medicine, Anthropology. Director of Obstetrics and Gynecology, Sydenham Hospital, New York, N. Y.

Paley, D. H., M.D., Biology. Research Assistant, Mt. Sinai Hospital, New York, N.Y.

ACTIVE MEMBERSHIP

Alimurung, Mariano M., M.D., Internal Medicine. Research Fellow, The House of The Good Samaritan, Boston, Mass.

Angle, William D., M.D., Internal Medicine. House Staff, Long Island College Hospital, Brooklyn, N. Y.

Appel, S. Baer, M.D., Internal Medicine. Assistant Resident, Bellevue Hospital, New York, N. Y.

Altman, Irving E., M.D., Veterinary Medicine. Veterinary, Private Practice, Brooklyn, N. Y.

Arai, Harold S., M.D., Internal Medicine. Resident in Cardiology, Mt. Sinai Hospital, New York, N. Y.

Basco, Daniel M., M.S., Geology. Professor of Geology, Legazpi Junior College, Legaspi, Philippines.

Bass, Allan D., M.D., Pharmacology. Professor and Chairman of Department of Pharmacology, College of Medicine, Syracuse University, Syracuse, N. Y.

Berens, Conrad, M.D., Opthalmology. Professor of Clinical Opthalmology, New York University, New York, N. Y.

Bizzozero, Orpheus, M.D., Endocrinology. Director of Education, Waterbury Hospital, Waterbury, Conn.

Broomfield, Elizabeth, B.S., Bacteriology. Bacteriologist, Control Laboratories, Burroughs Wellcome & Co., Inc.

Bruno, Leonard C., B.S., Embryology, Endocrinology. Graduate Assistant, De Paul University, Chicago, Illinois.

Clary, Mary L., A.B., Biology. Junior Pharmacologist, Ciba Pharmaceutical Products Inc., Summit, N. J.

Conrad, Lester I., B.S., Pharmaceutical Chemistry. Technical Director, American Cholesterol Products, Inc., Milltown, N. J.

Corman, Harvey H., M.D., Psychiatry. Research Fellow, Manhattan State Hospital, New York, N. Y.

Costa, Pasquale J., A.B., Pharmacology. Graduate Student, Yale University, New Haven, Conn.

Deckert, Evangeline F., M.A., Pharmacology. Director of Pharmacological Research, Reed & Carnrick, Jersey City, N. J.

Elster, Samuel K., M.D., Pathogenesis of Rheumatic Fever. Chief, Pathologic Chemistry, Army Medical Department, Research and Graduate School, AMC, Washington, D. C.

Galgan, Robert J., B.S., Botany. Graduate Assistant, De Paul University, Chicago, Illinois.

Gordon, Alvin J., M.D., Medicine. Research Assistant, Mt. Sinai Hospital, New York, N. Y.

Greenberg, Boris E., M.D., Human Fertility. Consulting Urologist, Beth Israel Hospital, Boston, Mass.

Gutmann, Helmut R., M.D., Biochemistry. Research Associate, Department of Chemistry, School of Biological Sciences, University of Tennessee, Memphis, Tenn.

Hall, Marion R., Ph.D., Clinical Psychology. Consulting Psychologist, Brooklyn Hospital, New York, N. Y.

Jeanloz, Roger W., Ph.D., Organic Chemistry & Biochemistry. Member, Worcester Foundation for Experimental Biology, Shrewsbury, Mass.

Kelly, John W., A.B., Biology. Research Assistant, University of Pennsylvania, Philadelphia, Pa.

Kiesewetter, Herbert F., B.S., Psychology. Designer, J. G. White, Inc., New York, N. Y.

King, Thomas J., M.S., Physiology, Biochemistry. Teaching Fellow in Biology, Washington Square College, New York University, New York, N. Y.

Klavano, Paul A., D.V.M., Pharmacology. Assistant Professor of Veterinary Medicine, State College of Washington, Pullman, Wash.

Kline, Paul R., M.D., Medicine. Assistant Clinical Professor, Bellevue Medical Center, New York, N. Y.

Kuta, Virginia A., B.S., Invertebrate Zoology. Graduate Assistant, DePaul University, Chicago, Illinois.

Lasagna, Louis, M.D., Internal Medicine. Resident Physician, Maimonides Hospital, Brooklyn, N. Y.

Levy, Guy W., Geology. Broker, Levy Brothers, New York, N. Y.

Loewy, Paul, M.D., Biology. Physician, Private Practice, New York, N. Y.

Loop, Anne, Ph.D., Public Health. Assistant Professor, Department of Physiology and Hygiene, Hunter College, New York, N. Y.

Lutterloh, Charles H., M.D., Biology. Practicing Physician, Hot Springs, Arkansas.

Ma, Roberta M., Ph.D., Antibiotics. Biochemist, BAIC, U.S. Department of Agriculture, Beltsville, Md.

Marelli, John A., B.S., Organic Chemistry. Student, Fordham University, New York, N. Y.

McFarren, Earl F., B.A., Biochemistry. Research Assistant, National Dairy Research Laboratories, Inc., Oakdale, L.I., N. Y.

Megibow, Samuel J., M.D., Cardiovascular Surgery. Clinical Assistant, Mt. Sinai Hospital, New York, N. Y.

Mount, David T., B.A., Virology. Assistant Instructor, Animal Disease, University of Connecticut, Storrs, Conn.

Murphy, John B., Ph.D., Biological Science. Dean, Professor of Biology, St. Louis Preparatory Seminary, St. Louis, Missouri.

Nealen, Joseph P., M.S., General and Modern Physics. Head, Physics Department, Gonzaga University, Spokane, Wash.

Nelson, Dorothy K., Ph.B., Internal Medicine. Student, Long Island College of Medicine, Brooklyn, N. Y.

Nerad, Josephine J., B.S., Invertebrate Zoology. Graduate Assistant, De Paul University, Chicago, Illinois.

Pelcak, Emil J., B.S., Bacteriology, Chemistry. Micro-Biological Assay, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

Polnauer, Frederick F., M.E., Bio-Mechanics, Physics. Research Engineer, New York University, New York, N. Y.

Pordy, Leon, M.D., Internal Medicine. Research Assistant in Cardiology, Mt. Sinai Hospital, New York, N. Y.

Quinn, Robert W., M.D., Internal Medicine. Instructor in Preventive Medicine, Yale University, New Haven, Conn.

Reyes, Felix M., M.D., Biology, Chemistry. Pathologist, V. A. San Patricio Hospital, San Juan, Puerto Rico.

Robinson, Franklin, M.D., Neurology. Senior Resident, Neurology and Neurological Surgery, St. Vincent's Hospital, New York, N. Y.

Roger, Alex A., M.D., Internal Medicine. Adjunct, Cedars of Lebanon Hospital, Los Angeles, Calif.

Russ, Walter R., B.S., Pharmaceutical and Medicinal Chemistry. Research Director, Mycoloid Laboratories Inc., Little Falls, N. J.

Sachs, Bernard A., M.D., Internal Medicine. As sistant Resident in Medicine, Montefiore Hospital, New York, N. Y.

Schack, Jerome A., M.D., Biochemistry. Instructor, Army Medical Department, Research and Graduate School, Washington, D. C.

Schiaffino, Stephen, M. S., Vitamins, Nutrition. Biochemist, Vitamin Division, Food and Drug Administration, Washington, D. C.

Shaw, Ernest W., M.D., Infectious Diseases. Research Fellow, Department of Preventive Medicine, Yale University School of Medicine, New Haven, Conn.

Sherlock, Marjorie, B.A., Library. Chief Librarian, University of Alberta, Edmonton, Alberta, Canada.

Smyers, William H., LL.B., Chemistry and Anthropology. Patent Attorney, Standard Oil Company, Elizabeth, N. J.

Sonenberg, Martin, M.D., Thyroid Metabolism. Fellow, American Cancer Society, New York, N. Y.

Steinman, D. B., Ph.D., Engineering. Consulting Engineer, New York, N. Y.

Swift, Shelley A., M.D., Pathology. Resident Pathologist, Philadelphia General Hospital, Philadelphia, Pa.

Thorell, Bo, M.D., Cytochemistry, Hematology. Docent at Karolinska Institutet, Stockholm, Sweden.
Tucker, George S., Sc.D., Biology. Author-Lecturer, New York, N. Y.,
Usdin, Myron, M.S., Bacteriology. Graduate Assistant, Department of Bacteriology, Ohio State University, Columbus, Ohio.
Zimmerman, Irving, D.V.M., Biology. Practicing Veterinarian, New York, N. Y.
Zuckerman, Seymour, M.D., Internal Medicine. Private Practice, Brooklyn, N. Y.

STUDENT MEMBERSHIP

Aronow, Lewis, Biology. Student, College of the City of New York, New York, N. Y.
Hardy, Frank M., B.A., Biology. Student, New York University, New York, N.Y.
Lipkin, Lewis E., B.A., Medicine, Pathology. Student, Long Island College of Medicine, Brooklyn, N. Y.
McCarthy, John R., M.A., Organic Chemistry. Student, Columbia University, New York, N. Y.
Plummer, Jewel I., M.S., Cell Physiology. Teaching Fellow, New York University, New York, N. Y.
Pollak, Peter I., M.S., Chemistry. Student, Columbia University, New York, N. Y.
Pound, Guy M., M.S., Physical Chemistry. Assistant in Chemistry, Columbia University, New York, N. Y.
Schreiber, Kurt C., B.S., Chemistry. Ph.D. Candidate, Columbia University, New York, N. Y.

ADDITIONS TO LIFE MEMBERSHIP CLASS BY TRANSFER

Abel, Theodora A.
Adams, A. Elizabeth
Aeschlimann, John A.
Albaum, Harry G.
Applebaum, Edmund
Arnow, L. Earle
Atkin, Lawrence
Ball, Edwin L.
Bartelstone, Herbert J.
Bassett, Lucy W.
Baum, Sidney J.
Beale, Helen P.
Berger, Adolph R.
Bichler, David L.
Blumberg, Harold
Brown, Ellis V.
Caldwell, A. Lee
Carlson, Gustaf H.
Chevigny, Hector
Clarke, Beverly L.
Coykendal, Frederick
Crosman, Arthur M.
Crossley, M. L.
Daniel, Earle H.
Davis, Herbert L.
De Beer, Edwin J.
Denton, J. J.
Deyrup, Ingrith J.
Diamond, Moses
Dunning, William B.
Ebel, Robert
Ells, V. R.
Fahrenbach, M. J.
Ford, Mary F.
Fox, Jay T.
Gladwin, Thomas
Gordon, Samuel M.
Gurd, Frank R.
Gustav, Alice
Hanke, Harriett H.
Hickman, Kenneth C.
Hill, Henry E.
Hilton, James G.
Hughes, Edward C.
Hultquist, Martin E.
Jakowska, Sophie
Johann, Norma E.
Joyner, Austin
Killian, John A.
King, Cecil V.
Kittredge, M. B.
Kleinberg, William
Knobloch, Herbert J.
Lamb, Willis E., Jr.
LaMer, Victor K.
Larson, Nora L.
Lazier, W. A.
Levy, Martin W.
Light, Amos E.
Light, Richard U.
Light, Robert F.
Malcolm, Wilbur G.
Markham, Floyd S.
Marple, Charles D.
Martin, Earl A.
Mattin, Harry E.
Meyerding, Henry W.
Miner, Eunice T.
Mirand, Edwin A.
Morrison, A. Cressy
Mossman, Donald D.
Mushett, Charles W.
Nelson, Theodora
Niedercorn, Joseph G.
Nims, Leslie F.
Oser, Bernard L.
Parker, Robert P.
Reiss, Isidor
Rioch, David M.
Roduta, Feliciano L.
Roth, Barbara
Schiller, Joseph
Schroeder, Charles R.
Seeger, Doris R.
Sherwood, Marion B.
Stebbins, Robert B.
Stenger, Albert H.
Stokes, J. G. Phelps
Tee Van, John
Thompson, Henry D.
Thompson, Kenneth W.
Van Burkalow, Anastasia
Wainio, W. W.
Walcott, William W.
Weathers, Curtis L.
Weiner, Nathan
Whiteside-Hawel, Beatrice
Winkelman, Nathaniel W.